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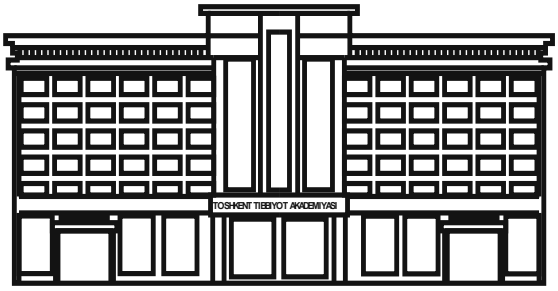

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MYOCARDIAL MORPHOLOGY ON THE BACKGROUND OF DIABETES

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The purpose of the study: to determine the nature of the morphological and morphometric changes of the heart myocardium layer in experimental diabetes.

Materials and research methods: To achieve the goal of the study, the heart of 56 sexually mature white laboratory rats was studied. White laboratory rats were divided into 3 groups. The first group consists of rats in the control group, mature intact rats. In our second group, after placing the laboratory rats in a special cage, we inject 4 mg of streptozotocin per 100 g of body weight into the abdominal cavity to induce experimental diabetes, and blood sugar levels are measured daily by taking blood from the tail vein. Rats are killed 30 and 60 days after the experiment. Tissues were taken from the heart for histological examinations. Heart tissue was fixed in 10% formalin solution, dehydrated in alcohol, and paraffin blocks were prepared. Histological preparations of 8-12 μm were prepared from the prepared paraffin blocks and stained by the hematoxylin-eosin method.

Results: The heart is a hollow muscular organ. It is the main member of the vascular system that moves blood. The heart and average body weight of white breed laboratory rats is 220±6.9 g. The heart of rats is bounded in the chest, on both sides by the lungs, on the lower side by the diaphragm, and on the back by the thoracic aorta. The results of the morphometric examination of the ventricles of the rat heart on the 30th day of the experiment showed that the thickness of the wall of all sections of the heart was smaller compared to the control group. Blood filling, stasis and diapedesis hemorrhages were detected in the small vessels of the heart, accompanied by their perivascular swelling, bulging and disorganization of the connective tissue stroma. In the subepicardial zone of the myocardium, it was found that there are enlarged and full vessels. Initial tumors were detected in the myocardial stroma. An increase in edema was observed in the myocardial stroma, mainly in the perivenular and pericapillary spaces. It was found that collagen fibers swell and signs of mild disorganization of connective tissue begin to appear. Blood vessels have a rounded shape due to endothelial cell swelling. Clear cytoplasmic fluid appeared in the cytoplasm of small vacuoles in cardiomyocytes. That is, the development of hydroptic dystrophy was observed in cardiomyocytes. Cardiomyocytes with dystrophic changes and unchanged cardiomyocytes can also be seen with a focal appearance of intracellular swelling. Signs of lymphohistocytic infiltration were also detected. On the 60th day of the experiment, the changes in the myocardial blood vessels were preserved, that is, the fullness of the veins, blood thinning, and perivascular hemorrhages in the form of numerous diapedesis were recorded. Intramuscular fluid has been observed to push muscle fibers. In the myocardial tissue, initially focal and then scattered infiltrates consisting of lymphocytes, histiocytes and fibroblasts were seen. Signs of mucoid and fibrinoid staining were detected in the myocardial stroma. Signs of protein hydropic dystrophy were observed in cardiomyocytes, swelling appeared inside the cell, and numerous foci of plasmolysis were detected in the myocardium. Areas of plasmolysis look like optical voids. Vacuolation can be seen in the cytoplasm of cardiomyocytes. Thickening and swelling of collagen fibers were found in the endocardium. Interstitial swelling increases and spreads throughout the myocardium. Swelling of the collagen fibers caused their fibers to shrink. Myocardial tissue swells and disorganization of surrounding connective tissue fibers is observed. Dystrophic changes of the myocardium have a diffuse character, absorption of the cytoplasm and increased intracellular edema were observed. Muscle fibers twitching and focal swellings were detected.

Conclusion. The results of the study showed that dystrophic changes are noted in the heart wall of rats with diabetes. According to our observations, the inflammatory process reached its maximum level on the 30th day of the study, signs of perivascular hemorrhages and interstitial edema, lymphohistocytic infiltration, mucoid and fibrinoid secretions were detected in the myocardial stroma. In the subendocardial area of the myocardium, a more pronounced edema was seen.

KETOGENIC DIET IN TREATMENT OF METABOLIC SYNDROME

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Introduction. Metabolic syndrome (MetS) is a cluster of conditions comprising central obesity, dyslipidemia, hypertension, and insulin resistance which has been shown to increase the risk of cardiovascular diseases and type 2 diabetes. The ketogenic diet (KD) has recently gained popularity as a potential treatment modality for MetS by virtue of promoting ketosis, which is an alternative metabolic state where the body derives its metabolism from fat utilization rather than carbohydrate utilization. Several studies have reported that KD leads to significant weight loss, improvement in insulin sensitivity, and favorable changes in lipid profiles. However, long term safety and adherence to such strict dietary regimen remains uncertain.

Methods. A systematic review and meta-analysis of keto diet effects on MetS were performed according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. We searched PubMed, Scopus, and Web of Science for studies published between January 2010 and March 2023. All clinical trials or observational studies reporting keto diet intervention in humans with MetS (IRB approval), which reported at least

one of the target outcomes: fasting glucose, HbA1c, HOMA-IR, triglycerides, HDL cholesterol, and CRP were included. Prospective animal study designs were not allowed. The inclusion criteria implied that 70–75% fat, 20–25% protein, and 5–10% carb was used. We excluded studies that lasted less than three months both prospective clinical trials or prospective observational studies from this meta-analysis.

Results. The review included 20 studies. KD led to mean reductions in body weight of 7.5% (6.0–9.0, $p < 0.01$), fasting plasma glucose of -15.0% ($p < 0.05$), HbA1c of -0.5% (-0.3–0.7, $p < 0.05$), insulin sensitivity of +25% (18-32, $p < 0.01$), triglycerides of -20% (−15 – −25%, $p < 0.01$) and an increase in HDL cholesterol of +10% (+5-15%, $p < 0.05$), and CRP levels declined by −30% ($p < 0.05$).

Discussion. The KD is effective in improving metabolic parameters in individuals with MetS. with significant weight loss (average 7.5%) comparable to previous studies, which further underlines the potential of KD for weight management to reduce risk for cardiovascular disease and type 2 diabetes. Also, KD reduces fasting glucose by 15% and HbA1c by 0.5%, indicating increased insulin sensitivity. Concurrently, KD decreases the triglyceride level by 20%, while increasing HDL cholesterol level by 10%, suggesting beneficial influences on lipid metabolism and cardiovascular risk factors. A 30% reduction in CRP, indicative of reduced systemic inflammation associated with cardiovascular diseases, was observed in the present study. However, similar to any intervention, the long-term sustainability and safety of KD warrants consideration. Continual adherence over time is a known challenge as well as monitoring for potential adverse effects such as nutrient deficiencies.

Conclusion. The KD appears to be effective in managing MetS by promoting weight loss, enhancing glycemic control, increasing insulin sensitivity, and improving lipid profiles, all while decreasing systemic inflammation. Nevertheless, more research is needed to ensure the long-term sustainability and safety of this diet, as well as to establish thorough clinical guidelines.

NON-ALCOHOLIC FATTY LIVER DISEASE AND ITS TREATMENT METHODS

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Introduction. Non-alcoholic fatty liver disease (NAFLD) is an epidemic liver disease characterized by excessive accumulation of lipids in the liver parenchyma. This disease is the most common chronic liver disease, and NAFLD currently affects approximately 20-30% of the world's population, but is 90% more common in obese people. NAFLD and metabolic syndrome share common pathophysiological mechanisms such as cardiovascular disease, chronic kidney disease, sleep apnea, obesity, insulin resistance, and diabetes. Non-alcoholic fatty liver disease is the liver manifestation of metabolic syndrome. Creating effective therapeutic strategies for the treatment of this disease is of interest to hepatologists. For this reason, a deep study of the pathogenesis of NAFLD and the creation of effective treatment methods is an urgent issue.

Methods. We searched PubMed, Scopus, and Web of Science for studies published between January 2019 and March 2024. All clinical trials or observational studies reporting dietary interventions in people with NAFLD reported at least the primary outcomes: body mass index; ALT, AST, HOMA-IR, triglycerides, HDL cholesterol. Physical activity (150 min per week) and a diet low in saturated fat and processed foods (1700-2400 kcal per day) and moderate caloric restriction (1200-1800 kcal per day) were used. Studies that included patients with concurrent liver disease or for whom excessive alcohol consumption was not examined were excluded.

Results. The review included 20 studies. Traditional and Mediterranean (modified) diet and physical activity significantly reduced weight, waist circumference, body fat percentage, greater than 7% weight loss significantly improved steatosis, higher liver enzymes decreased, liver lipids decreased by 13%, lipid oxidation, glucose and IR improved. Metformin and pioglitazone were taken when using drugs. ALT and AST levels and insulin sensitivity improved in the first 3 months. No improvement was observed after 3 months. When pioglitazone was discontinued, ALT increased, adiponectin decreased, insulin sensitivity worsened, and liver fat increased, with no change in fibrosis.

Discussion.NAFLD has a bidirectional relationship with components of the metabolic syndrome and increases the risk of type 2 diabetes, cirrhosis, and related complications. Because of the complex pathophysiology and significant heterogeneity of disease phenotypes, combination therapy may be required for many patients with NAFLD. A healthy lifestyle and weight loss are crucial in the prevention and treatment of NAFLD. In this sense, weight loss has been shown to reduce the risk of cardiovascular disease and diabetes, as well as reverse liver disease and improve fibrosis. In addition, regular exercise in people with NAFLD has been shown to reduce intrahepatic fat, induce hepatoprotective autophagy, increase fatty acid b-oxidation, and upregulate peroxisome proliferator-activated receptor-g (PPAR-g). may ameliorate NAFLD through various mechanisms, including increasing insulin sensitivity and reducing hepatotension. Apoptosis. There is currently no approved drug therapy for NAFLD, although several drugs are in advanced development because adherence over time is diet a known problem, as well as possible adverse effects such as nutrient deficiencies.

Conclusion. The increase in NAFLD has and will continue to burden the health care system, especially because of its ties to obesity, IR and metabolic syndrome. Currently, the understanding of its epidemiology and pathogenesis are well understood, guidelines for proper care are constantly changing as new information emerges, but still

Содержание